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RYEGRASS BLAST

Cattle production in the southern United States increased from 1964 to 1973, decreased during 1974–1982, and then stabilized to almost one-half of the total herds in the 48 contiguous states. Success of the livestock industry in the region depends on the availability of quality forages for grazing and hay. The large number of animals and a favorable environment make year-round grazing a desirable goal.

Ryegrass is one of the most important forage crops grown in the southern United States (23). Perennial ryegrass (*Lolium perenne* L.) and annual or Italian ryegrass (*L. multiflorum* Lam.) are the most economically important species in the genus *Lolium*. Mild winters favor ryegrass production because plants are less winter-hardy than other cool-season forage species, with only minimal growth during periods of extended low temperatures. Annual ryegrass is nutritious and palatable and produces excellent weight gains in beef cattle when compared with other winter pastures. Annual ryegrass also may be interseeded in row

crops in the fall or planted as a cover crop to reduce soil erosion. In the spring, winter annuals may be treated with herbicides and the resultant sod planted to corn, soybeans, cotton, or summer annual grazing crops. Currently, about 8 million hectares in the southern United States are managed as annual pastures.

The most important diseases on ryegrass in the United States include crown rust (8), ergot (8,23), cottony blight (30), blind seed disease (8,23), and leaf spots (8,23). In the fall of 1971, a previously unreported disease on ryegrass was observed by Bain (5) in Mississippi and Carver et al (9) in Louisiana. The disease reached epidemic proportions and reduced ryegrass forage yields in both states (5–7,9,24). Destruction of ryegrass forage was so severe that the disease became known as ryegrass blast (9). The disease has become endemic in these areas, and localized outbreaks occur every year, although the outbreaks have not been as widespread as or caused the economic losses that occurred during the 1971 epidemic. The annual threat of a ryegrass blast epidemic necessitates a greater understanding of the pathogen and the factors that govern disease development.

Symptom Development

Initial symptoms are small brown spots or gray-green, water-soaked spots on the leaves and stems of ryegrass seedlings. The spots develop into round or oval lesions with gray or blue-gray centers and dark brown or purple margins. A chlorotic border surrounds

older lesions (Fig. 1). Gray-white to reddish brown lesions are concentrated at leaf tips. Bain et al (7) and Carver et al (9) describe overall symptoms as a blast or blight, with plants appearing flaccid, water-soaked, and dark green. In the field, patches of severely diseased plants result in thick mats of dead ryegrass (Fig. 2).

A common production practice in Mississippi and Louisiana in 1971 was to interplant small grains with ryegrass (6,24). These small grains also developed blast symptoms, but losses were minimal (24). Oats interplanted with ryegrass were not significantly affected by blast (Fig. 3) (5,6). Symptoms on ryegrass interplanted with oats differ from those on ryegrass seeded alone and include severe water-soaking and large areas of necrotic tissue; discrete lesions are difficult to discern on these plants. A rank odor of freshly decomposed plant tissue is noticeable in interplanted fields (5,6). These symptoms are thought to result from the larger oat plants decreasing aeration in the plant canopy and soil surface. Enhanced activity of soil microorganisms in this environment may result in rapid death and decomposition of the ryegrass (5–7).

The Causal Organism

Magnaporthe grisea (T.T. Hebert) Yaegashi & Udagawa (anamorph = *Pyricularia grisea* (Cooke) Sacc.) was consistently associated with diseased plants in the 1971 epidemic (Fig. 4) (5–7,9,24). Koch's postulates were completed in subsequent greenhouse

Fig. 1. Blast lesions on individual ryegrass leaf; chlorosis borders older lesions.

Fig. 2. Field symptoms of blast include thick mats of dead ryegrass and an odor of freshly decomposed plant tissue.

Fig. 3. Blast has killed all Gulf ryegrass plants in a mixed planting with Florida 501 oats; only oat plants are left standing.
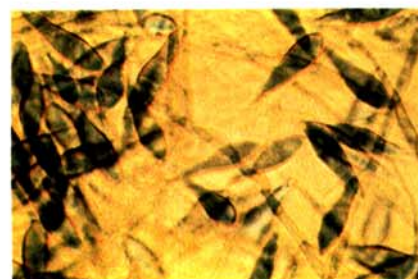

Fig. 4. Characteristic three-celled conidia of *Magnaporthe grisea* stained with cotton blue.

Fig. 5. Symptoms of *Magnaporthe grisea* infection of (A) crabgrass, (B) pearl millet, and (C) St. Augustinegrass.

inoculation tests, and *M. grisea* was established as the causal agent of ryegrass blast (5–7). Prior to 1971, natural infection of *Lolium* species by this fungus had not been reported (9), although artificial infection of both annual and perennial ryegrasses was reported in Japan in 1956 (20).

The fungus has a wide host range that includes corn (*Zea mays* L.) (4), crabgrass (*Digitaria sanguinalis* (L.) Scop.) (12) (Fig. 5A), pangola grass (*D. decumbens* Stent) (13), pearl millet (*Pennisetum glaucum* (L.) R. Br.) (21) (Fig. 5B), oats (*Avena sativa* L.) (5,7), St. Augustinegrass (*Stenotaphrum secundatum* (Walter) Kuntze) (12,19) (Fig. 5C), wheat (*Triticum aestivum* L.) (7,29), and rice (*Oryza sativa* L.) (31). These and other plant species in the host range of the fungus occur in Mississippi and Louisiana and may be important reservoirs of inoculum.

Conidia of *M. grisea* penetrate leaf tissue directly by forming appressoria or enter through stomates (12). Haustoria are produced after penetration and invade mesophyll cells.

Epidemiology

Because blast had not been detected on ryegrass in Mississippi or Louisiana prior to 1971, the origin and mechanism of dissemination of the pathogen in ryegrass production areas were unknown. Ranganathaiah and Mathur (22) showed that seed can disseminate *M. grisea*. However, the widespread distribution of blast in Mississippi and Louisiana suggested that dissemination by seed was not a major factor in initial disease development (5,6). Spores of *M. grisea* are readily disseminated by wind (17). Hurricane Edith reached the Gulf shores of Mississippi and Louisiana from the Caribbean on 15 and 16 September, only a few days before noticeable symptoms developed on ryegrass (5–7). Bain et al (7) questioned that an epidemic of this severity could be initiated by the dispersal of inoculum in such a short time. They concluded that the fungus was already present and that environmental conditions associated with Hurricane Edith were ideal for spore production and disease development. Rain, fog, and heavy dew associated with the hurricane helped accelerate blast development (5–7).

Research on other diseases incited by *M. grisea* confirms that high moisture is a contributing factor in disease development (12,19). In Guadeloupe, French West Indies, moisture on the foliage of pangola grass in the form of dew or rain is essential for infection, with maximum foliar damage occurring during the wet season (13). Isaac and Owen (12) found *Magnaporthe* infection of St. Augustinegrass in Florida shortly after heavy rains in early spring. The low incidence of gray leaf spot on St. Augustinegrass in southern California has been attributed to infrequent rain and low humidity (19).

For infection of rice leaves by *M. grisea*, leaf wetness periods of 6–16 hours are required at temperatures of 18–32 C

(14,15). Asai et al (3) reported that a considerable number of lesions were produced when dew was relatively heavy for 8 hours or longer but that few lesions were produced when dew was absent or persisted for less than 8 hours. When the quantity of dew was small, even though the dew period was 8 hours or longer, the number of lesions produced was generally fewer than when the dew period was comparable length but the amounts of dew were greater. Andersen et al (2) found that 16–24 hours of high humidity and continual wetness were required for maximum infection of rice at 22–28 C and that inoculated plants kept in a humidity tent for 10 hours or less did not become infected. They concluded that since most infections were established in 21.5 hours, exposure to continual wetness for an equal or greater length of time would be necessary for maximum secondary spread of inoculum and establishment of an epidemic in the field.

Although rain and high humidity are common conditions necessary for disease development, Suzuki (25) felt that temperature was the most important single factor in development of rice blast. Several researchers have reported that the optimum temperature for infection of rice is between 22 and 29 C (2,14, 15,26). This temperature optimum was often based on the requirement for 16–24 hours of dew or leaf wetness (2,14). Rice seedlings grown at night temperatures between 15 and 20 C were more susceptible than plants grown at night temperatures of 26 C or higher (16). However, Asai et al (3) found no statistical correlation between temperature and lesion number when night temperatures were between 18 and 28 C and day temperatures were between 23 and 25 C.

Age of plants and time of infection have been suggested as important factors in the development of ryegrass blast (5,6, 9,24). Small seedlings up to 1 month old were rapidly killed, whereas older, more mature plants showed only leaf spots and leaf necrosis (5,6). Rice plants responded similarly in the seedling and tillering stages but became resistant in the booting stage (2,14). Abe (1) found that the greatest number of blast lesions on rice plants in the three- to five-leaf stage occurred on medium-sized leaves. The middle of the leaf had the greatest number of lesions, and the leaf tip showed more symptoms than the base. On older leaves, the base had more lesions than the tip. In leaves of rice plants grown in the greenhouse during winter months, susceptibility to infection was greatest in 1- to 4-week-old plants and decreased as plant age increased to 10–11 weeks; leaves of plants 11–24 weeks of age were resistant (14).

Choice of cultivar may also be implicated in epidemics of M. grisea (26). In 1971, most fields of ryegrass in Louisiana and Mississippi were planted with the cultivars Gulf, Magnolia, Wintergreen, and Common. These cultivars proved to be susceptible to ryegrass blast (5–7), and producers suffered heavy economic losses of winter forage for cattle (24).

We investigated the effects of cultivar, plant age, inoculum density, temperature, leaf wetness duration, planting date, and reduced solar radiation on infection by M. grisea and disease development, with the following results:

1. Although Gulf ryegrass was susceptible in all stages of development, lesion numbers increased with plant age until 4–5 weeks, then began to decrease (Fig. 6). A cubic polynomial regression equation, which accounted for 88% of the variation within the data, predicted that 33-day-old plants were most susceptible (18). When 3-week-old plants were exposed to inoculum densities of M. grisea increasing up to 8.0×10^5 conidia per milliliter, total lesion numbers increased exponentially on the second, third, and fourth youngest leaves.

2. Blast symptoms did not develop on ryegrass plants maintained at very low or very high temperatures. A few lesions were found on plants held at 35 C, but no lesions developed on plants maintained at 5 C. A cubic polynomial model represented the best-fit regression equation for temperature data (Fig. 7). This
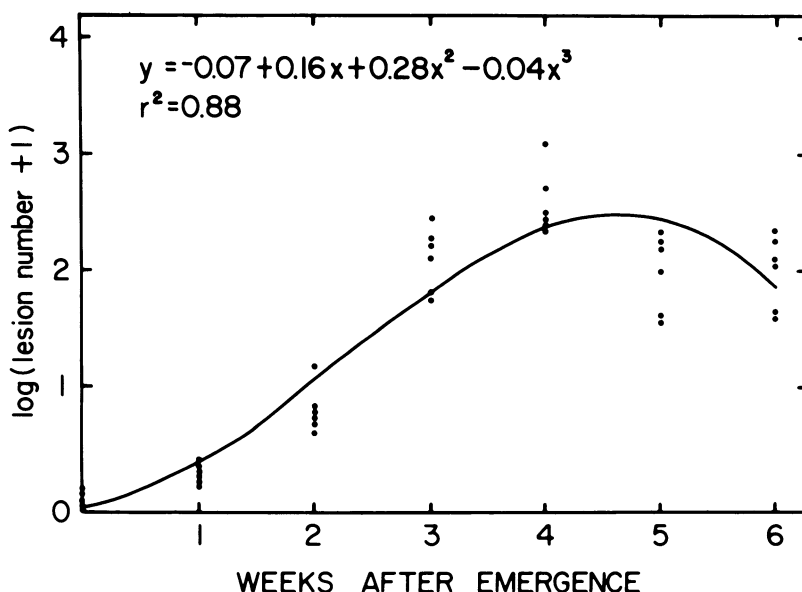
$$y = -0.07 + 0.16x + 0.28x^2 - 0.04x^3$$
$$r^2 = 0.88$$

Fig. 6. Relationship of lesion number to increasing age of Gulf ryegrass plants inoculated with *Magnaporthe grisea* at 2.0×10^5 conidia per milliliter. (From Moss and Trevathan [18])

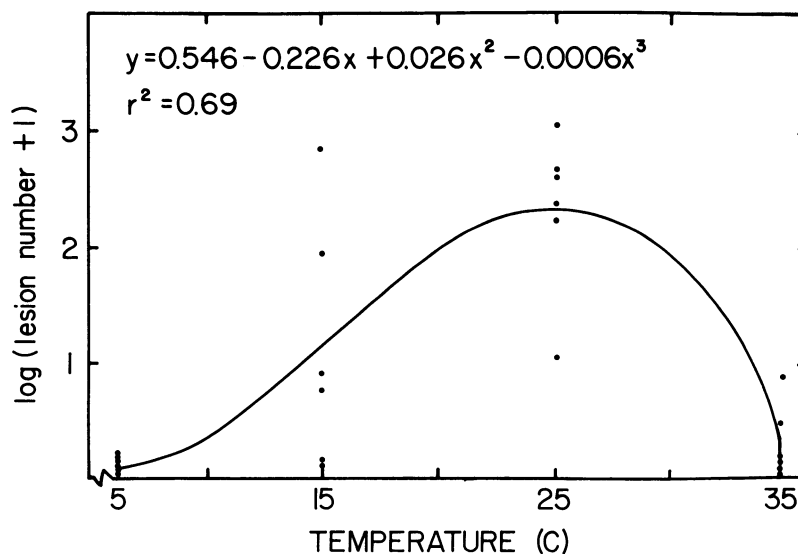$$y = 0.546 - 0.226x + 0.026x^2 - 0.0006x^3$$
$$r^2 = 0.69$$

Fig. 7. Relationship of lesion number on 3-week-old Gulf ryegrass plants to incubation at different temperatures for 72 hours following inoculation with *Magnaporthe grisea* at 2.0×10^5 conidia per milliliter. (From Moss and Trevathan [18])

model resulted in a coefficient of determination of 0.69 and predicted 26 C to be the optimum temperature for *M. grisea* infection of 3-week-old Gulf ryegrass.

3. Free moisture on leaf surfaces was essential for infection. Lesion numbers increased exponentially with increasing periods of free moisture up to 24 hours. Continual wetness periods beyond 24 hours did not significantly increase the number of lesions produced during one disease cycle.

4. Gulf ryegrass was more susceptible than Marshall, Sunbelt, or Tetrablend 444 to infection by *M. grisea*. These cultivars produced an average of 3.20, 0.12, 0.13, and 0.05 lesions per plant, respectively, when inoculated with 2.0×10^5 conidia per milliliter and incubated for 72 hours at 27 C (18).

5. Delayed planting of ryegrass decreased disease incidence when plants were inoculated at 3 weeks and leaf wetness was maintained for 96 hours. The number of lesions per 40 g of leaf tissue was 128, 72, 6, and 1 for Gulf ryegrass planted on 15 September, 1 October, 15 October, and 1 November, respectively. Exposing plants to increasing amounts of shade also reduced disease incidence.

Response of Plant Introductions

To determine the response of ryegrass plant introductions to artificial inoculation under greenhouse conditions, we evaluated 315 plant introductions from 41 countries (28). Symptoms in plants subjected to consecutive inoculations ranged from no lesions to plant death, and the plant introductions most resistant to *M. grisea* were from Europe. Eight introductions were asymptomatic for ryegrass blast, and these, as well as most of the 123 introductions that survived, were perennials. Only 20 of 82 annuals tested and one perennial $\times$ annual cross were among the survivors. The eight sources of possible immunity to one isolate of *M. grisea* infecting ryegrass have also shown field resistance to other ryegrass pathogens and adaptation to environmental conditions in Mississippi.

Characteristics of Isolates

Because isolates of *M. grisea* are reported to be variable, we compared isolates and their relative pathogenicity (27). The fungus was obtained from various hosts throughout Mississippi. Six isolates were cultured from symptomatic leaf tissue of crabgrass and three isolates from symptomatic St. Augustinegrass. Single isolates were obtained from asymptomatic soybean (*Glycine max* (L.) Merr.) pod tissue and from lesions on leaf tissues of spurge (*Euphorbia preslii* Guss.), smartweed (*Polygonum pensylvanicum* L.), and Italian ryegrass. Colony morphology

depended on both isolate and growth medium. Growth rate also varied among isolates.

All isolates, except one from crabgrass, incited visible symptoms on one or both of the ryegrass cultivars Gulf and Magnolia (27). Small, water-soaked spots on leaves became round to oval lesions with gray centers 4 days after inoculation. Symptom development was most obvious 5 days after inoculation, and lesions were concentrated on adaxial leaf surfaces. Lesions often coalesced, extending the width of leaf blades and killing the leaf from that point to the leaf tip. Dieback of leaves was also observed in association with older lesions bordered by chlorosis. Chlorosis tended to expand from the area of the original lesion toward the leaf tip.

Virulent isolates were generally gray to white, whereas black isolates were weakly virulent (27). Culture appearance varied from abundant white aerial mycelium to very sparse appressed growth. The more virulent isolates generally grew the fastest, but no definite relationship was established between colony growth rate and virulence. Although disease varied with the isolate tested, symptom expression and disease development were the same regardless of inoculum source.

Source of the 1971 Strain

The source of the strain of *M. grisea* that incited the ryegrass blast epidemic in 1971 is unknown. Because rice culture is widespread in Louisiana and Mississippi, it is possible that the rice blast fungus infected ryegrass. However, recent studies have shown that this strain of the fungus is no more likely to be responsible for infection of previously unreported hosts than strains from other hosts or other geographical areas (29). The fact that isolates of the fungus from a number of hosts were able to infect ryegrass suggests an absence of inter-strain barriers. This, along with an active parasexual cycle, represents likely sources of variation in nature for *M. grisea*.

Significant meteorological events, such as hurricanes, represent sources of introduction of fungal strains and vehicles of widespread movement. In addition to the presence of a virulent strain, specific environmental conditions and host-pathogen relationships are necessary for epidemics of ryegrass blast. Environmental conditions associated with mesoscale weather events were not previously considered predictive because of the random component in local circulation characteristics that spawned those events. There are now seasonal hurricane predictive signals for the Atlantic basin from global and regional predictors (10). Such predictive relationships are not operative or are much weaker in other tropical cyclone basins. The two extended-range predictors for Atlantic

tropical cyclones identified to operate on a long time scale are the strength of stratospheric quasi-biennial oscillation zonal wind near 10° north latitude and rainfall in West Africa.

In a summary of statistics for Atlantic tropical cyclones over the last 40 years, the number of mesoscale weather events was highest, and the intensity of those events greatest, during a 20-year period including the 1950s and 1960s (11). This same pattern was also true for the total number of hurricane days and the number of intense-category hurricane days involved in those events. All four parameters declined during the 10-year period ending in 1980 and have gradually increased since then. The fact that the ryegrass blast epidemic occurred in association with Hurricane Edith at the end of the most active hurricane period in the past 40 years should be considered in any retrospective analyses to develop predictions of risk years. Meanwhile, the recommendation to delay fall planting may reduce total forage availability from fall to early summer (18), but it does prevent total losses of forage such as those experienced in 1971.

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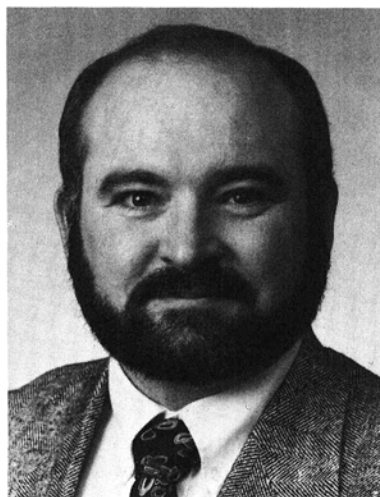
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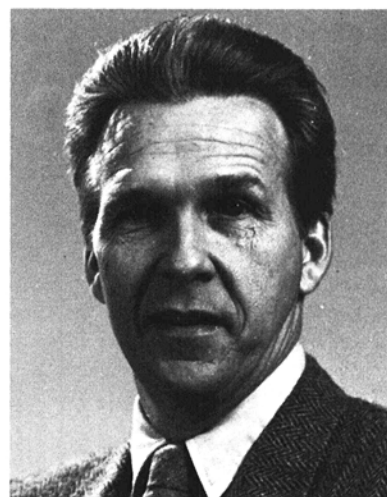
Don Blasingame

Dr. Blasingame retired from Mississippi State University in 1991 after many years of service as an extension plant pathologist. He received the B.S. degree in biology and chemistry from Delta State University and the M.S. degree in plant pathology from Mississippi State University. He earned the Ph.D. degree in plant pathology from Texas A&M University. Dr. Blasingame served as an assistant and as an associate extension plant pathologist at Mississippi State, where his responsibilities included development of educational programs for the control of diseases and nematodes of cotton, small grains, and forage crops. Before his retirement, he authored more than 100 extension publications. He currently is director of technical services for the Southern Turfgrass Association.